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Oxygen Consumption During Repeated Inertial Load Exercise Sprints with Differing Recovery Durations in Older Adults

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Oxygen Consumption During Repeated Inertial Load Exercise Sprints with Differing Recovery Durations in Older Adults

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Dedication

This Report is dedicated to anyone who keeps showing up even when no one else thinks you can or will; life is a race and always chase the faster pace.

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Abstract

Oxygen Consumption During Inertial Load Exercise Sprints with Differing Recovery Durations in Older Adults

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As the generation termed the "baby boomers" continue to age the proportion of older adults in the population continues to climb increasing the demand for viable ways to improve "health span" by exercise to improve cardiovascular and musculoskeletal health and reduce medical costs due to unhealthy aging. Inertial load cycle ergometer (ILE) sprints lasting 4s each, have been shown to provide consistent maximum power outputs in individuals of all ages. This study examined the effect of 60s, 45s, and 30s recoveries between sprints, performed for a total of 15 minutes, on oxygen consumption and heart rate. Six subjects aged 61 ± 2.6 yrs and VO_{2peaks} of 2.91 ± 0.51 L/min completed three separate trials of 15 ILE sprints with 60s recovery, 20 ILE sprints with 45s recovery, and 30 ILE sprints with 30s recovery during weeks 1, 4, and 8 respectively of a progressive eight-week repeated ILE training program that consisted of three training sessions per week. During each trial 6 of the ILE sprints included VO_2 collection using breath by breath, average of total power, HR, and RPE were recorded across similar time points during each trial. Percentage of VO_{2peak} was measured at 38.22 ± 2.17, 61.3 ± 6.50, and 59.14 ± 5.31

during the trials of 60s, 45s, and 30s recoveries between sprints respectively (60s recovery vs. 45s recovery p=0.0138; 60s recovery vs. 30s recovery p=0.0251). %HR maximal was not statistically different between trials (p=0.133). Within the population there was a correlation between average power maximums and VO_2 (r=.73; p=0.0006), VO_2 and estimated stroke volume ($SV_{Est.}$) (r=0.94; p <0.0001), and rating of perceived exertion (RPE) and %HR maximal (r=0.24; p<0.05) during each ILE sprint trial. In conclusion, ILE sprints may elicit the same maximal power outputs with 60s, 45s, and 30s recoveries with comparable cardiovascular stress following 45s and 30s recovery periods.

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RESEARCH AND HYPOTHESES

This study examined the effect of 60s, 45s, and 30s recoveries between sprints, performed for a total of 15 minutes, on oxygen consumption and heart rate. The specific hypothesis was as follows:

- Different recovery times between repeated ILE sprints in older adults would result in a significant difference in oxygen uptake and heart rate responses during an eight-week progressive repeated ILE sprint program. We hypothesized that cardiovascular stress, measured by oxygen consumption and heart rate, would increase as recovery duration decreased during ILE sprint trials of 60s, 45s, and 30s between sprints.

REVIEW OF LITERATURE

Introduction

During team sports events, unlike endurance events, athletes will undergo repeated sprints of durations lasting anywhere from 4-12s in duration with short recoveries followed by longer recoveries multiple times throughout entire matches (Girard et al., 2011). These athletes do not have low aerobic capacities, however, with junior level hockey players having VO_{2max} levels reported between 56-65ml/kg/min (Montgomery, 2006), suggesting a developed aerobic component despite heavy and possibly exclusive emphasis on solely repeated sprint training. Most of the literature on repeated sprints (duration less than 6s) has not focused on the aerobic training effect; and to our knowledge, no studies have been conducted assessing repeated sprint ability in older populations, who may benefit the most from such training given the associated decreases in both duration as well as intensity of exercise with age (Goodpaster et al., 2006). The purpose of this review shall be to describe repeated sprint ability and its effects on aerobic capacity with respect to metabolism compared to traditional programs and the potential direction for research on this topic related to the potential benefits for older adults.

Section I: Repeated Sprint Ability and Metabolic Factors

How the term "sprint" is defined and used varies depending on context. To accurately assess repeated sprint ability (RSA), for the purposes of this review, a sprint will be considered an all-out effort of a duration of less than 10s (Girard et al., 2011). Anything greater than 10s and the effort produced will not be able to be sustained at a peak ATP turnover rate, becoming more submaximal in nature, although it should be noted others will refer to longer durations for sprints. Protocols for field-based settings may implement repeated sprints with work to rest ratios varying from 1:3 to 1:30, creating a spectrum for repeated sprint ability (McLellan, Lovell, & Gass, 2011; M. Spencer, Bishop, Dawson, & Goodman, 2005; M. Spencer et al., 2004). Thus, repeated sprint protocols can be delineated further into intermittent sprints and repeated sprints, depending on work to rest ratios with the former defined as having a work to rest ratio of greater than 1:6 and the latter considered anything less than 1:6, suggesting that intermittent sprints should show reduced fatigue while repeated sprints should yield greater fatigue with the reduced recovery times, resulting in differing levels of stress and reliance on the energy system (Girard et al., 2011).

While types of exercise are often divided into "aerobic" or "anaerobic" according to which processes the required energy is primarily being attained, all exercises use all processes to some degree to meet the energy demands of the task at hand. With respect to repeated sprint protocols, work to rest ratio as well as sprint duration dictate the proportions of those demands. Gaitanos et al. performed a study where eight male volunteers performed 10x-6s repeated sprints with 30s recoveries (1:6 work to rest ratio) on a frictionally braked cycle ergometer and found that anaerobic glycolysis contributed to 44% of the energy production in the first sprint compared to 16% on the final sprint, showing

how energy system contribution levels shift to more oxidative pathways as fatigue increases, as there was a 47.5% decrease in power from the first to the tenth sprint (Gaitanos, Williams, Boobis, & Brooks, 1993). How RSA is developed and utilized becomes a question of the specific goals following sprint based programs, necessitating an understanding of the energy pathways and their relationships.

All energy for muscular contraction is a byproduct of the hydrolytic breakdown of adenosine triphosphate (ATP) into ADP and P_i. Intracellular ATP at peak turnover rates of 15 mmol/kg/dm/s are able to support maximal work for only 1-2s before becoming exhausted (Glaister, 2005). Once depleted a reliance on the ability to resynthesize ATP via P_i from the phosphocreatine (PCr) pathway becomes the limiting factor in the ability to produce energy and thereby work during repeated maximal sprints. Substantial evidence is lacking in what signaling pathways result in PCr pathway upregulation during repeated sprints, but currently the prevailing theory is that intracellular ADP concentration increases are responsible (McMahon & Jenkins, 2002). Much like intracellular ATP at the initial onset of exercise, PCr has a peak turnover rate of roughly 9mmol/kg/dm/s lasting roughly 1-2s, making the PCr system ideal for supporting short high intensity activities, particularly if resynthesized quickly (Bogdanis, Nevill, Boobis, & Lakomy, 1996; McMahon & Jenkins, 2002; M. Spencer et al., 2005) .

While the oxidative energy pathway provides relatively smaller proportions of total energy (3-8%) during an initial sprint, that contribution quickly increases as recovery time drops or the number or duration of sprints increases (de Aguiar, Turnes, Santos de Oliveira Cruz, Salvador, & Caputo, 2015; M. Spencer et al., 2005). During a full Wingate test roughly 17% of total energy was produced via oxidative metabolism compared to 44% following a second full Wingate test with 4minutes of recovery (1:4 work rest ratio) (Gaitanos et al., 1993). In a different study by Balsom et al. subjects performed repeated

sprints of 15, 30, and 40m, matching for total sprint volume, with maximal oxygen consumption following the last sprint significantly lower following the final 15m sprint compared to both the 30m and 40m sprints, suggesting an increased demand for oxidative metabolism as sprint duration increases even when total sprint volume remains constant (Balsom, Seger, Sjodin, & Ekblom, 1992). Intuitively, a greater oxidative capacity during scenarios with greater metabolic stress will be more advantageous as less demand is required of limited absolute PCr stores. Decreased glycolytic strain has the other added benefit of decreased metabolite buildup, allowing for more stable intramuscular conditions (Brooks, 2009; Enoka & Stuart, 1992).

In the same study by Gaitanos et al. of 10x 6s repeated cycle ergometer sprints, PCr contributed to 49% of the total ATP for the first sprint and 80% for the final sprint (Gaitanos et al., 1993). While total energy production dropped, as indicated by the power decrements across the 10 sprints, the reliance on PCr contribution increased, emphasizing the importance of PCr in energy production during repeated sprints. Interestingly, lactate levels were also found to be the same during the first and tenth sprints suggesting that aerobic pathways aid in the maintenance of power during repeated sprints (Brooks, 2009; Casey, Short, Curtis, & Greenhaff, 1996; Gaitanos et al., 1993). Given that fatigue of the muscles is what ultimately results in the inability to produce maximal force it is important to know what is causing the fatigue, and for repeated sprints how to limit the amount of resistance to such fatigue.

Section II: Repeated Sprint Ability and Fatigue Indices

When comparing the muscle contractions required of cycling versus running during repeated sprints, there is a favorable advantage to cycling due to the reduced eccentric load compared to during running sprints (Enoka & Stuart, 1992). Maximal concentric contractions are less difficult to generate from the central nervous system (CNS) compared to eccentric contractions as a byproduct of differences in motor unit recruitment patterns (Nardone, Romano, & Schieppati, 1989). To better understand whether the ability or inability to maintain maximal force in repeated sprints is a byproduct of a physiological inability to produce maximal power or a byproduct of other neuromuscular or psychological factors, studies assessing force contractions followed by electrical stimulation have been conducted (Bigland-Ritchie, Johansson, Lippold, & Woods, 1983; Bigland-Ritchie, Kukulka, Lippold, & Woods, 1982; Hales & Gandevia, 1988). In studies conducted where muscles were repetitively stimulated at submaximal levels muscle force generation declined over time with little to no changes in glycogen, lactate, or ATP (Hamm et al., 1988; Hamm, Reinking, & Stuart, 1989; Jami, Murthy, Petit, & Zytnicki, 1983). A proposed reason for this is that there is a potential decrease in the sensitivity of the contractile machinery resulting in a decreased ability to generate force despite adequate stimuli being applied and substrate availability for force production (Enoka & Stuart, 1992). A possible mechanistic explanation for this excitation contraction desensitization involves a reduction of the Ca^{2+} reuptake into the sarcoplasmic reticulum (Allen, Lee, & Westerblad, 1989; Enoka & Stuart, 1992; Westerblad & Lannergren, 1991). In the context of repeated maximal sprints such mechanisms of fatigue would likely necessitate larger numbers of sprints than found in most studies, but could become more relevant as duration increases.

When it comes to fatigue and RSA the greatest correlation appears to be between peak power and fatigue (Girard et al., 2011). The closer that an individual is to their maximum power the greater the speed at which subsequent sprints appear to fatigue (Bishop, Spencer, Duffield, & Lawrence, 2001; Gaitanos et al., 1993; Girard et al., 2011). This in line with the observation that an endurance runner is capable of running several VO_{2max} intervals consistently with minimal recovery whereas a sprinter may run one high level sprint with either a high level of recovery required or large power decrements in subsequent sprints (Bishop & Spencer, 2004). The example just provided comes back to the difference in fiber type recruitment and the primary energy pathways that those fiber types produce energy when recruited. High force is likely produced by more fast glycolytic and intermediate fibers which lack mitochondrial density and consume high levels of ATP whereas endurance activities are likely more reliant on mitochondrially dense slow oxidative fibers (Bishop & Spencer, 2004). For RSA, the former are most pertinent and, due to decreased mitochondrial density, are limited in the ability to clear and handle the metabolic load and its resulting metabolite buildup over time.

As alluded to earlier, PCr availability correlates with RSA, being a critical predictor of fatigue during repeated sprints (Girard et al., 2011). Obtaining valid PCr levels post sprint though becomes difficult as accurate assessment of PCr requires muscle biopsies which ideally would need to be performed immediately following the completion of a sprint or series of sprints. Regardless of the raw values, however, it is apparent that individuals who have lower PCr stores exhibit lower force production during sprints or power decrements following PCr depletion (Bogdanis, Nevill, Boobis, Lakomy, & Nevill, 1995; Girard et al., 2011; Sahlin & Ren, 1989). Added support for this is that creatine supplementation has been found to decrease the rate of fatigue between repeated sprints (Kreider, 2003). Yquel et al. found that 6 days of 20g of creatine resulted in an increased

power output of 5% and increased muscle PCr during a repeated sprint protocol (Yquel, Arsac, Thiaudiere, Canioni, & Manier, 2002). If a valid ergogenic aide, in the case of team sports, creatine supplementation could provide an edge if athletes are able to outsprint competitors to make important plays. Additionally, creatine has been shown to buffer inorganic phosphate limiting inhibition of contractile coupling machinery by potentially decreasing the signaling of phosphorylase *a* and phospho-fructose kinase and the otherwise consequent upregulations of creatine kinase, which may improve lactate clearance and thereby RSA (Brooks, 2009; Parolin et al., 1999). The benefits of creatine supplementation during RSA is not universal though with studies finding that there is no correlation between creatine supplementation and RSA (McKenna, Morton, Selig, & Snow, 1999). The association between PCr and RSA has been rationalized as being a result of potential differences in fiber types of subjects since PCr levels are favorably higher at rest in type II fibers compared to type I fibers (Demant & Rhodes, 1999; Glaister, 2005). One study reported a PCr content of 73.1 ± 9.5 and 82.7 ± 11.2 mmol/kg/dm, which would suggest that there might be a preponderance of "responders" and "non-responders" following creatine supplementation (Demant & Rhodes, 1999). In a study where 10s maximal sprints were performed by subjects, biopsies indicated a 45% decrease in PCr in type I fibers from baseline compared to 62% in type IIa fibers from baseline, further suggesting a greater reliance on PCr utilization dependent on fiber type (Karatzaferi, de Haan, van Mechelen, & Sargeant, 2001). Further research and efforts to tease out these confounding factors with creatine supplementation are necessary as well as a greater standardization of supplementation protocols in creatine RSA studies conducted.

Given that glycogen is a primary fuel source during glycolytic work it makes sense to presume there should be a relationship between repeated RSA and glycogen storage. A cross-over design study by Spencer & Katz looked at the influence of glycogen depletion

preceding two 95% of VO_{2peak} tests with glycogen supercompensation and glycogen depletion (M. K. Spencer & Katz, 1991). As expected, the glycogen depletion resulted in a lower peak power output compared to the group with supercompensation, but the fatigue between the two bouts was not significantly different, suggesting that the rate of glycogenolysis is independent of glycogen content (M. K. Spencer & Katz, 1991). Additionally, the production of lactate has been found to be similar in groups irrespective of glycogen baseline levels, suggesting similar glycolytic rates (Bangsbo, Graham, Kiens, & Saltin, 1992). So unless at a drastic depleted state at baseline, it can be concluded that intramuscular levels of glycogen are not a rate limiting factor in RSA (Parolin et al., 1999; Ren, Broberg, Sahlin, & Hultman, 1990).

Of the metabolites that are built up during repeated sprints, H^+ ions produce increased levels of acidosis which influence the ability of muscles to produce the appropriate levels of ATP (Enoka & Stuart, 1992). Brooks et al. estimates that ATP hydrolysis standard free energy can drop to as low as -11kcal/mol in exercising muscle produced acidic conditions (Brooks et al. 2005). How well this relates to fatigue is questionable though since high power has been seen in conjunction with high H^+ levels suggesting that acidosis is likely not to be the leading cause of fatigue during RSA, particularly since it has been shown in several studies that pH does not recover as quickly as power output (Glaister, 2008). The original study on acidosis and skeletal muscle fatigue was also conducted originally in rabbit muscle at acidic levels not feasible in humans and since has been replicated with non-repeatable findings further suggesting an over emphasis of H^+ influence on fatigue in human exercise (Dawson, Gadian, & Wilkie, 1978). While acidosis may not be a direct cause to fatigue, lactate in the blood which is produced parallel to the drop in pH intracellularly does play a potential role in the cardio-autonomic regulation of variables such as sympathetic nerve activity, heart rate, and blood

pressure measured during or following repeated sprints via activation of skeletal muscle group IV afferents (Kaufman & Hayes, 2002; Mitchell, Kaufman, & Iwamoto, 1983).

Unlike acidosis, P_i accumulation has now become the believed cause of fatigue during high intensity activity (Dahlstedt, Katz, & Westerblad, 2001; Dahlstedt & Westerblad, 2001; Girard et al., 2011). In semitendinosus muscle of frogs, peak force correlated with P_i levels and has been found to play a role in the inhibition of Ca^{2+} ion reuptake to the sarcoplasmic reticulum which would result in contractile coupling dysfunction, an issue which has been previously addressed (Fitts, 2008; Thompson & Fitts, 1992). Supporting this notion, a study looking at subjects without creatine kinase, a key enzyme, exhibited no decrease in Ca^{2+} sensitivity following force production (Allen, Kabbara, & Westerblad, 2002; Allen & Westerblad, 2001; Dahlstedt & Westerblad, 2001). However, while a lack of creatine kinase results in a decrease in observed fatigue, a large reduction in power output compared to individuals who have creatine kinase is observed likely due to the inability to utilize the PCr system effectively in absence of creatine kinase.

What mode of recovery that is implemented during repeated sprints appears to also affect the level of fatigue experienced in RSA protocols. Some studies have shown that while implementing recovery at rates as low as 20-30% of VO_{2peak} between repeated sprint bouts of 6s in duration, RSA improves compared to with passive recovery between sprint bouts (M. Spencer et al., 2006). Proposed rationales for this are increased metabolite clearance and potential fuel enhancement as a byproduct of recycled lactate via the Cori cycle (M. Spencer et al., 2005). Given that most RSA settings are in team sports settings where active recovery is almost always necessary, this is a practical method of potential RSA improvement during competition.

Important to consider as well is which mode of exercise is implemented when assessing RSA in subjects as specificity of sport plays a large role even when considering

what type of cycle ergometer to use rather than just whether cycling or running protocols or implemented (Girard et al., 2011). For instance when considering isokinetic cycling compared to frictionally loaded cycling the decrease in performance is a byproduct of decreased force in isokinetic training versus decreased velocity in frictionally loaded cycling (Bishop et al., 2001; Girard et al., 2011). How well these measurements transfer to the goals, whether that be general fitness or performance, of everyday application requires further exploration.

Section III: VO_{2max}, Sprint Intervals, and Future Research

While the majority of this review has assessed RSA in relation to the mechanistic aspects of fatigue development and fuel utilization in ways most pertinent to individuals engaging in RSA for the purpose of performance, the remainder of this review will be addressing how RSA may be a viable alternative to traditional methods of cardiorespiratory exercise with particular attention to VO_{2max} with respect to general health and fitness.

Exercise receives fuel from both oxidative and glycolytic pathways as discussed, with RSA relying primarily on glycolytic pathways but is enhanced by a strong oxidative capacity and metabolic system. The oxidative capacity of an individual is largely dependent or limited by the maximal oxygen consumption that an individual can utilize by large muscle groups during maximal steady state exercise otherwise known as VO_{2max} or VO_{2peak}. Important to note is that glycolytic pathways are still utilized during exercises that emphasize oxidative metabolism. A. V. Hill's lab is credited with having first quantified the relationship between VO_2 and HR responses, with heart rate having a linear relationship with increased VO_2, while additionally defining the concept of oxygen deficit, where the oxygen consumption is delayed at exercise onset compared to the demands required for a given intensity (Bassett 2002). VO_{2max} can be measured by assessing the difference between oxygen inspired compared to that expired in a given gas sample over a quantified period of time usually expressed as liters per minute during cycling or relative to body mass as $ml/kg/min^{-1}$ while running. These early studies made use of meteorological balloons for gas collection with the technological expansion of breath by breath analyzers in the late 1960s and 1970s allowing for a greater development of testing and analysis of VO_2 (Poole & Jones, 2012).

Given that cardiorespiratory fitness has been measured by VO_{2max} for approximately 100 years, it is not surprising that it has been studied extensively (Bassett & Howley, 1997; Green & Patla, 1992; Howley, Bassett, & Welch, 1995; Poole & Jones, 2017). VO_{2max} has been shown to increase through the adolescence, peaking in early adulthood with a 1% decline on average for every year after 30 or 40 (Dehn & Bruce, 1972; Fleg & Lakatta, 1988; Hagberg, 1987; Heath, Hagberg, Ehsani, & Holloszy, 1981; Rogers, Hagberg, Martin, Ehsani, & Holloszy, 1990). Endurance training however has been shown to increase VO_{2max} in previously untrained individuals with increases as high as 23% (Hickson, Hagberg, Ehsani, & Holloszy, 1981). Much of the increase in VO_{2max} following training varies with respect to training history and the baseline VO_{2max} of the individuals being trained. Studies show that those individuals with already high VO_{2max} values or high training loads are unlikely to see increases in VO_{2max} after a point. Coyle and Joyner have written a review on the determinants of endurance performance in greater detail that goes beyond the scope and purpose of this review (Joyner & Coyle, 2008). Previously, it was also believed that individuals undergoing endurance training programs could be separated into groups of "responders" and "non-responders" but a recent study by Montero and Lundby split subjects into varying groups of training volume and those who were considered non-responders following an increased training load were converted from non-responders to responders (Montero & Lundby, 2017). While endurance training may only improve VO_{2max} in individuals up to a point, training has also been shown to reduce the declines associated with aging when intensity and volume are maintained (Heath et al., 1981; Lanza et al., 2008). Training is reversible however with declines in VO_{2max} beginning as soon as one week after cessation of training, with as much as 40% of training level $VO2_{max}$ lost after 4 weeks of detraining, and after 16 weeks a full return to pretraining VO_{2max} levels (Coyle, Hemmert, & Coggan, 1986; Coyle et al., 1984). More recently,

research has taken a greater interest in older adult populations due to the increased role of aging in society, building on findings from earlier studies which have shown VO_{2max} can be increased when training programs are started later in life or maintained at higher levels in spite of aging, reaching numbers comparable to untrained and some moderately trained young adults when training intensity and duration are high enough (Huang, Gibson, Tran, & Osness, 2005; Rogers et al., 1990).

It is well documented that as individuals age beyond their twenties the level and intensity of physical activity declines gradually and that increases in sedentary behavior bring increased mortality risk (Ekelund et al., 2016; Sallis, 2000; Telama & Yang, 2000; Trost et al., 2002). With these decreases come decreases in cardiorespiratory fitness as well as skeletal muscle health and function (Goodpaster et al., 2006). Muscles are recruited according to the size principle such that type I fibers are preferentially recruited first, requiring lower intensities and frequencies, and before the recruitment of type IIa and type IIx fibers, which require higher frequencies and intensities of neural drive (Milner-Brown, Stein, & Yemm, 1973). Thus, with lower intensities and overall activity levels a paucity of type II fiber type recruitment is likely a contributing factor to both decreased overall strength as well as atrophy of overall muscle mass with progressing age. Type II fibers exhibit an increase in cross sectional area with training compared to type I fibers which exhibit a decrease in cross sectional area with training; both adaptations serve to facilitate the function of specificity in the structure-function relationship (Fitts & Widrick, 1996).

During endurance training programs VO_{2max} increases as a result of improvements of one or both parts of the Fick equation (VO_2 = Cardiac Output * a-vO_2 difference), cardiac output or a-vO_2 difference, with overall stroke volume at any given exercise intensity increasing as a result of left ventricular wall mass increases, increased capillary

to muscle fiber ratio allowing for improved oxygen delivery to the exercising muscle, increased mitochondrial density, and an increased overall blood volume (Holloszy, 2011; Holloszy & Coyle, 1984; Meredith et al., 1989; Saltin et al., 1976). While effective there are diminishing returns with volume in that VO_{2max} increases will taper off over time once central adaptations have reached their maximum (Gore, Hahn, Burge, & Telford, 1997).

When volume has reached a maximum, variations in intensity become more important for adaptations and increases in intensity may be necessary for continued improvements. In a classic study by Hickson et al. untrained subjects underwent 9 weeks of a high intensity program alternating between days of doing VO_{2peak} intervals on a cycle ergometer and running 30min as hard as possible on a motorized treadmill with one rest day per week where a VO_{2max} test was conducted to assess training adaptations and ensure progressive overload during the VO_{2peak} cycling intervals (Hickson et al., 1981). A total of 23% improvement in VO_{2max} following the training program was observed (Hickson et al., 1981). The major dilemma with such a program, while effective, is the mental and physical stress that it induces. When asked if the subjects would be willing to continue a follow-up study continuing the training, most subjects declined due to the high demands of the training required (Hickson et al., 1981). Studies have shown that when the training load is matched between moderate intensity and high intensity exercise that VO_{2max} increases to the same extent requiring considerably less time (Gibala, 2007; Gibala et al., 2006; MacInnis & Gibala, 2017).

In the last two decades extensive research has looked at the potential benefits of engaging in higher intensity shorter duration intervals in lieu of traditional endurance training programs for the sake of saving time and increasing exercise participation (Gibala, 2007; Gibala et al., 2006). These shorter intensity intervals being explored range primarily from 30s to 4min in duration and require an intensity that would elicit VO_{2max} or greater

and have been categorized as sprint intervals or cumulatively as sprint interval training (MacInnis & Gibala, 2017). In one study subjects trained one leg on a cycle ergometer with moderate continuous exercise and the contralateral leg with sprint interval training with workloads matched for training load and it was found that mitochondrial density was improved to a greater extent in the contralateral leg than in the moderate continuous exercise trained leg (MacInnis et al., 2017). When normalized for oxidative capacity however, the mitochondrial function remained unchanged (MacInnis et al., 2017).

When engaging in higher intensity exercise such as sprint interval training compared to moderate continuous exercise there is a greater amount of relative physiological stress. Due to the higher level of ATP turnover within a given time frame there is a greater increase in metabolite and free radical buildup resulting in an increased activation of signaling pathways for proteins such as AMP-kinase, resulting in upregulation of gene expression for PGC-1α, the leading transcriptional co-factor for mitochondrial biogenesis, leading to greater mitochondrial content following sprint interval training (Holloszy, 2011; MacInnis & Gibala, 2017). With increases in skeletal muscle adaptations due to sprint interval training there should be an expected increase in overall oxidative capacity or VO_{2max}. In a study by Burgomaster et al. where subjects underwent a 6-wk training protocol of either traditional moderate intensity continuous training (40-60min) or sprint interval training (4 all out 30s Wingate tests) both groups exhibited similar increases in VO_{2peak} following the 6-wk training program (Burgomaster et al., 2008).

While research has shown that individuals who engage in continuous moderate intensity exercise and high intensity sprint interval training of 30s or longer in duration have similar improvements in cardiovascular fitness and skeletal muscle adaptations there have been fewer studies showing the effect of repeated sprints of less than 30s duration with respect to cardiorespiratory fitness, particularly repeated sprint programs

(Burgomaster et al., 2008; Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005; Gibala & McGee, 2008; MacDougall et al., 1998). If repeated sprint programs could be implemented and yield similar responses to sprint interval training, individuals who are looking to improve their cardiorespiratory fitness with limited time and limited exercise capacities could be greatly benefited.

STUDY: OXYGEN CONSUMPTION DURING REPEATED INERTIAL LOAD ERGOMETER SPRINTS WITH DIFFERING RECOVERY DURATIONS IN OLDER ADULTS

Introduction

In the industrialized world of today one of the greatest issues to contend with is aging and the rapid increase in the world population of those who are older than 65 years of age. In developed nations the purported numbers may reach more than 40% of the population (Petsko, 2008). Much of this increase is considered to be a byproduct of both the technological advances in medicine as well as the large birth rates reported during the baby boomer era contributing to increased life expectancy rates and decreased birth rates. Complicating this increase in life expectancy, older individuals are cardiovascular diseases leading to decreased function with increased health care costs, estimated in some cases to be already at a burden of close to $7 trillion dollars globally (Goldman et al., 2013). Recent efforts have been promoted in hopes of combatting these costs by improving what has been coined "health span" or the number of years without disfunction and disease within the life span of individuals (Kirkland & Peterson, 2009; Seals, Justice, & LaRocca, 2016). Given that a number of studies have begun to show that individuals who exercise at moderate to vigorous intensities while maintaining overall healthy life styles show prolonged health span (Beeson, Mills, Phillips, Andress, & Fraser, 1989; Hitt, Young-Xu, Silver, & Perls, 1999; Willcox, Willcox, & Ferrucci, 2008), and that those individuals who participate in excessive inactivity have been shown to exhibit increased risk of mortality (Ekelund et al., 2016) a push for greater cardiovascular exercise prescription and adherence is necessary. Currently the American College of Sports Medicine recommends that individuals

accumulate 150min of moderate or 75min of vigorous intensity exercise weekly to reap the minimum benefits and cardioprotective benefits of exercise. Getting individuals to adhere to these guidelines, however is another issue of concern.

Traditional aerobic exercise is normally conducted at moderate prolonged intensities of between 60-70% of VO_{2max} while cycling, swimming, rowing, or running for durations of 30-90min most days of the week with changes primarily to central adaptations (MacDougall and Sale 2014). This type of training has been shown to result in increases in VO_{2max}, the gold standard for cardiorespiratory fitness and aerobic capacity. While effective, a critical complaint has been the time commitment and monotony of traditional programs, which lead to the development and implementation of high intensity interval training (HIIT) which has been found to have a greater influence on peripheral adaptations than traditional continuous moderate exercise (Davies, Packer, & Brooks, 1981; Gibala, 2018; Gibala & Hawley, 2017; MacInnis & Gibala, 2017). HIIT is characterized by intervals of alternating work and rest of 2-10min in duration at intensities ranging from 80-100% of VO_{2max} and has been shown to be just as effective as traditional aerobic exercise training programs in increasing VO_{2max} (MacInnis & Gibala, 2017). While traditional HIIT cuts down on the time commitment, further attempts have been made to decrease this component of training while still maintaining the cardiovascular benefits. As such, studies utilizing Wingate and modified Wingate protocols have been tested and have shown similar improvements in VO_{2max} as expected from traditional methods of aerobic exercise training (Gibala, Gillen, & Percival, 2014; Gillen et al., 2016). Studies assessing bouts shorter than 20-30s often have been conducted in the assessment of sprint performance ability in team sport athletes and not necessarily in the context of aerobic performance or cardiorespiratory health (Bishop, Girard, & Mendez-Villanueva, 2011; Girard, Mendez-Villanueva, &

Bishop, 2011; M. Spencer, Bishop, Dawson, Goodman, & Duffield, 2006). As such our lab conducted a study examining the effects of 4s ILE sprints with differing recovery durations, assessing the effect of differing recovery durations on oxygen uptake and heart rate. Given that no research has assessed repeated sprint ability in older adults, the study aimed to address this gap in the literature on repeated sprints.

Methods

Research Participants

Six healthy, untrained, older adults (4 males, 2 females) aged 61 ± 2.6 years were recruited from a cohort of a larger study to participate in the experiment. All subjects were considered physically healthy with no pre-existing health conditioning, being free of overt heart disease, hypertension, knee or hip joint risks, and current ongoing medications had no contraindications for exercise. Subjects had body mass of 81 ± 7.2 kg, height 175 ± 4.5cm, and body fat percentage 29.5 ± 2.5. None of the subjects were partaking in systematic exercise outside of the training sessions during the duration of the study. All subjects performed the same progressive 8-week training program on an inertial load cycle ergometer (ILE). Subjects had the purpose of the study and associated written informed consent discussed and completed prior to participation in the study. All procedures were approved by the IRB of the University of Texas at Austin before commencing the study.

Exercise with Diagnostic ECG

To screen for the presence of overt coronary heart disease (CHD), subjects who fit with the ACSM exercise testing requirement underwent a diagnostic 12-lead ECG at rest and during incremental exercise to 85% of maximal heart rate prior to inclusion in the study. Subjects performed an incremental protocol of 10-15W every 2min until either 85% of age predicted maximal heart was achieved or the supervising physician required the subject to stop. The ECG was monitored continuously, and BP and ratings of perceived exertion were recorded each minute. Only those subjects demonstrating no signs or symptoms of CHD were allowed to participate in the study. None of the subjects in this study failed to pass the ECG testing initially.

Training

After demonstrating no signs or symptoms of heart dysfunction as determined by the university physician, subjects were cleared for participation. Subjects performed a VO_{2peal} test on an electronically braked cycle ergometer (Lode ergometer, Groningen, The Netherlands) to assess maximal cardiorespiratory capacity. All subjects were partaking in an eight-week progressive training program of repeated sprints on an ILE three times a week. Each session consisted of a lower body warm-up of 10-20 body weight squats and lunges followed by repeated sprint training on the ILE according to the respective week and progression of training over the course of 8 weeks. For each sprint the subject was asked to cycle as hard and as fast as possible, lasting 3-4s , increasing pedaling cadence throughout the duration of the sprint. Verbal encouragement, as well as feedback of decreases and increases in power were provided to help illicit maximal efforts from subjects during each sprint. Training progressed from 15 repeated ILE sprints with 60s recovery during week 1, to 20 repeated ILE sprints with 45s recovery, to 30 repeated ILE sprints with 30s recovery. During one training session at the end of each section of the training progression (Week 1, Week 4, and Week 8) subjects' VO_2 data were collected during 6 ILE sprints. The selected sprints were between ILE sprints 10-20, depending on the respective section of training, allowing for a steady state to be achieved. Heart rate (Polar, USA) and rating of perceived exertion (RPE) on a numeric Likert scale from 6-20, with 6 being very easy and 20 being very, very, hard, were collected during every fifth ILE sprint during training sessions where VO_2 was collected for analysis (Borg et al., 1987).

Breath by Breath VO_2 Collection

Subjects breathed through a mouth piece attached to a two-way non-rebreathing valve connected to a pneumotachometer with VO_2 being analyzed via gas samples collected by a 6-foot capillary tube connected directly to a mass spectrometer (MA Tech Services, St. Louis, MO, and Beck Integrative Physiology Systems; Hans Rudolph, Kansas

City, MO company name), which provided O_2 and CO_2 concentrations during inspiration and expiration. The mass spectrometer was calibrated via a 2-point calibration using both room air and gas from a cannister of known measured concentration and the pneumotachometer was calibrated for volume using a 3-liter syringe at low, moderate, and high flow rates.

VO_{2peak} Testing

Subjects were fitted for the appropriate seat height on an electronically braked cycle ergometer where they were then familiarized with the mouth piece and nose clip at the appropriate height. Each subject was provided with a heart rate monitor across the chest to determine the heart rate progression through each stage as well as maximal heart rate at the conclusion of the test. Prior to initiating the test subjects were told to pedal at a cadence over 60 revolutions per minute with a preference of between 80-90 revolutions per minute. Protocols were tailored to each of the subjects with the goal of lasting between 8 and 12 minutes in duration. Estimated power outputs at VO_{2max} were estimated from age predicted heart rates and heart rates elicited during the submaximal stress test under medical supervision. Intensity was increased in a step-wise function by 10-20W, depending on the subject, every minute with heart rate and RPE collected during the last 10s of each stage and constant breath by breath collection of inspired and expired gases via the mass spectrometer. Tests were concluded when the subject either dropped below 60 revolutions per minute or voluntarily concluded the test. Subjects were verbally encouraged and motivated to give their best possible power outputs. Maximal heart rate was then recorded as the highest valued heart rate achieved during the test and used for future calculations.

Inertial Load Cycling Ergometry (ILE)

By use of a modified Monark (Varberg, Sweden) ergometer (Model 818) measurements of cycling torque and power during a 4s maximal sprint using only the

inertia of the wheel provided by the moment of inertia during flywheel acceleration was used to assess the power maximum during the entirety of the study. The exact specifications can be referred to elsewhere from previous studies in our department (Martin, Farrar, Wagner, & Spirduso, 2000; Martin, Wagner, & Coyle, 1997).

Dual X-Ray Absorbance Analysis (DXA)

DXA scans were run by trained technicians in the Fitness Institute of Texas at the University of Texas at Austin to assess body weight, fat mass, and fat free mass. For greater information on DXA refer elsewhere (Binkovitz, Henwood, & Sparke, 2008).

Statistics

$\%VO_{2peak}$ and %HR maximal values during 60s, 45s, and 30s recovery trials were assessed by an ordinary one-way ANOVA analysis. %HR maximal values at sprint 5, 10, 15, 20, 25, and 30, where applicable, were analyzed in each of the 60s, 45s, and 30s trial groupings by a repeated measures one-way ANOVA with an effect for time. Average power maximums during all 6 of the measured sprints for each ILE sprint trial for each subject were paired with VO_2 worked during each of the trials of 60s, 45s, and 30s recovery; %HR maximal was paired with RPE; estimated stroke volume was paired with VO_2. Pairs were assessed by linear regression models assessing correlations between paired variables. Subject characteristics, VO_{2peak}, age, height, weight, body fat%, and %HR maximal and VO_{2peak} from 60s, 45s, and 30s trials, were assessed using column statistics. Criterion for level of significance for each analysis was set at $p < .05$. All statistics were performed by the statistical software package Prism 7 Graphpad.

Results

Breath by Breath Oxygen Consumption

Subjects characteristics can be found in Table 1. Subjects VO_{2peak} at baseline was measured at 2.91 ± 0.50 L/min. $\%VO_{2peak}$ was measured at 38.22 ± 2.17, 61.30 ± 6.50, and 59.14 ± 5.31 L/min during the 6 ILE sprints of each trial of 60s, 45s, and 30s recoveries between sprints respectively. $\%VO_{2peak}$ was significantly different between the 60s recovery trial compared with both 45s ($p < 0.05$) and 30s ($p < 0.05$) trials respectively (Fig. 1). There was no significant difference between 45s and 30s recovery ($p > 0.05$) (Fig. 1). For VO_2 values for subjects during each trial refer to Table 2.

Correlations of Measurements and Power

Assessment of the relationship between absolute VO_2 and the average power maximum during the 6 measured sprints during each trial showed a strong correlation ($r=0.73$; $p < 0.05$) (Fig. 9). There were no significant differences between trials for power maximum averages ($p > 0.05$) (Fig. 8). For average power maximums produced by each subject during each trial refer to Table 3. The relationship between %HR maximal and RPE was positive and weak ($r=0.24$; $p < 0.05$). Estimated stroke volume values showed a strong linear correlation with VO_2 during each of the ILE sprint trials ($r=0.94$; $p < 0.0001$) (Fig. 6).

Percentage of Heart Rate Maximal

Comparison of %HR maximal measurements during the 60s recovery showed no significant differences between any time points ($p > 0.05$) (Fig. 2). %HR maximal measurements during the 45s recovery trial showed significant differences between sprint

5 and sprints 15 ($p < 0.05$) and 20 ($p < 0.05$) (Fig. 4B.). %HR maximal measurements during the 30s recovery trial showed significant differences between sprint 5 and sprints 20 ($p < 0.05$), 25 ($p < 0.01$), and 30 ($p < 0.001$) and between sprints 10 and 25 ($p < 0.05$) and 30 ($p < 0.05$), as well as sprint 25 and 30 ($p < 0.05$) (Fig. 5B.). All heart rate measurements took place at every fifth sprint and were recorded as the peak heart rate achieved during the recovery phase of the sprint as there was a delay in heart rate increase. For %HR maximal values for each subject during each of the trials refer to Table 4.

Estimated Stroke Volume

Using calculated values for O_2-pulse (VO_2/HR) estimated values of stroke volume for each trial were obtained. There were no statistically different values in stroke volume between ILE sprint trials of differing recoveries of 60s, 45s, and 30s (Fig 8.)

Discussion

The primary finding of this study was that during ILE sprints of 4s duration VO_2 values were equivalent to roughly 40% of VO_{2peak} at the minimum with 60s recovery and roughly 60% of VO_{2peak} with both 45s and 30s recovery between ILE sprints. To our knowledge this was the first study to explore the use of ILE sprints in an older population of individuals. Interestingly, while there was a significant increase in the elicited VO_2 response when transitioning from 60s to 45s recovery durations, there were no significant difference in VO_2 responses following the transitions from 45s to 30s recovery between sprints ($p > 0.05$). A possible explanation for this lack of increased VO_2 response was a lack of power increase between trials. While there was no statistical difference in the average power maximum produced between trials ($p > 0.05$) there was a strong correlation between average power maximum attained during the 6 sprints and the VO_2 elicited during each session (Fig 3b.). %HR maximal was also not significantly different between groups ($p > 0.05$), collectively suggesting that a potential lack of significant differences was a byproduct of not enough subjects in the study. Notwithstanding, the presence of trends towards significance cannot be neglected and potentially a training effect was preventing significance due to the progressive nature of the protocol in this study.

When looking at the potential cardiovascular effects of ILE sprints on older individuals it is interesting to note that heart rate continued to trend upwards in almost all subjects during all sessions (Fig. 3) (Fig. 4) with significance only in the 30s recovery session (Fig. 5). Part of the reason for potential differences in heart rate response can be attributed to varying degrees of initial fitness level, maximal heart rates, hydration levels,

and day to day stress levels. The sessions with 30s recovery between sprints showed the greatest development of %HR maximal during repeated ILE sprints (Fig. 5) with a significant difference between sprint 25 and 30 ($p < 0.05$) indicating a continuous increase in heart rate which may have continued to rise with further increases in sprint number and protocol duration (Coyle & Gonzalez-Alonso, 2001). Both the trials with 60s (Fig. 3B) and 45s (Fig. 4B) recovery did not show significant differences between time points with respect to heart rate, suggesting a shift in the mechanism of cardiovascular stress but both sessions did not have the same number of sprints and occurred at different time points within the training program, so caution should be taken when extrapolating conclusions. An additional point to consider is that all VO_2 measures were obtained in the middle of the training sessions ranging from sprints 10-15 during the 60s recovery session and sprints 15-20 in the 45s and 30s recovery sessions, meaning that possibly VO_2 did increase in the 30s session between sprints 25 and 30 compared to the other sprints, but was not measured. This is potentially still not the case however since subjects had reached a relatively steady VO_2 during each trial. Thus, another potential explanation for the greater rise in heart rate without a measurable increase in VO_2 was that metabolite buildup (e.g. P_i, H^+) resulted in an increased exercise pressor reflex response due to increased group IV afferent stimulation. Lactate measures during and following sessions would have provided better insight into this discrepancy in data.

Given that it is well established that RPE correlates with heart rate during steady state exercise and the subjects were exercising at a steady VO_2 during each of the trials, the low correlation value observed ($r=0.24$; $p < 0.05$) between RPE and %HR maximal

observed was potentially due to issues with pacing of the subjects as well as inexperience with providing maximal efforts in general v(Borg, Hassmen, & Lagerstrom, 1987; Green et al., 2006; Wenos, Wallace, Surburg, & Morris, 1996). Some of the subjects had no competitive sports backgrounds or have not engaged in competitive sports in decades and thus may not have been familiar with what would be defined as a maximal effort. Soriano-Maldonado et al. showed that older subjects who are provided a learning protocol for RPE show improvements in the relationship between heart rate and rating of perceived exertion, which may be more difficult for assessing sprints (Soriano-Maldonado et al., 2014). Given that the sprints were only 4s in duration and the load was progressively less as the sprint occurred coupled with the potential mix-match of efforts, it is not surprising that there was a lower correlation between %HR maximal and RPE. Another issue is that subjects may unconsciously or consciously have paced themselves knowing that there were many sprints to be completed in any given session, which was often confirmed by higher power maximum values in the last sprint compared to all other sprints. Verbal encouragement and power score feedback following each sprint were provided following each sprint in hopes of mitigating this issue, but motivational tactics cannot exclude pacing from having been a possibility. As a result, some subjects may have been capable of exercising at higher intensities during training sessions than what was performed.

Without invasive methods readily available non-invasive methods with strong correlations were important, particularly when assessing cardiovascular physiology. In this study an established model for estimating changes in stroke volume (SV) during each of the trials was used (Crisafulli et al., 2007; Whipp, Higgenbotham, & Cobb, 1996). Given

that in normal individuals a-vO_{2diff} does not always change in individuals following training and that there is a relatively constant value of 0.2 for a-vO_{2diff}, an estimated SV can be obtained using the equation:

$$SV_{Est.} = 5 \text{ X } \Delta VO_2/\Delta HR$$

where 5 is a constant, VO_2 is the amount of oxygen consumed at a given time point, and HR is the heart rate at that same time point (Whipp, Higgenbotham, & Cobb, 1996). This allowed for a more complete analysis of the Fick equation,

$$VO_2 = (SV*HR) \text{ X a-}vO_{2diff}$$

and potential assessment of what was causing VO_2 changes during each of the trials. However, given that our analysis showed no significant difference between trials for $SV_{Est.}$ ($p > 0.05$), it cannot be ruled out that shifts in heart rate or unaccounted-for a-vO_{2diff} were responsible. When looking at the correlation between $SV_{Est.}$ and VO_2 during each trial, there was a strong correlation ($r=0.94$; $p < 0.05$), which suggests that there was a VO_2-HR linear relationship across trials where measured. Future studies should assess whether the VO_2-HR relationship during ILE sprints differs from what is expected during more traditional steady state linear prediction models.

Limitations

This study was conducted as an ancillary exploration to a larger study that prevented randomization of order; an effect of order cannot be excluded as a confounding variable. Given that each trial occurred during a progressive training model, it cannot be excluded that an effect of training created the significant differences observed during the 60s recovery trial compared to 45s and 30s recovery trials. The conduction of tests in the morning versus the evening may have influenced the ability of subjects to elicit maximal power during all sessions depending on the demands of their lives and schedules. Differences in technicians who calibrated equipment can also not be excluded as a confounding variable. A larger number of subjects would have potentially allowed for some trends to become more significant and others less and should be considered in future study designs. Future studies should address the issues that have been already addressed in this study to better evaluate the effect of ILE sprints on VO_2 and the potential of ILE sprints as a viable alternative to traditional aerobic and power training modalities.

Conclusion

In conclusion, ILE sprints appear to elicit the same maximal power outputs with 60s, 45s, and 30s recoveries with the $\%VO_{2peak}$ being significantly elevated with 30s and 45s recovery compared to 60s recovery. Despite the fact that the sprints lasted only 4s, $\%VO_{2peak}$ was increased to approximately 60% VO_{2peak} with 30 or 45s recovery, demonstrating that maximal power training can moderately stimulate aerobic metabolism when the recovery period is short which may prove beneficial for populations who cannot engage in traditional exercise programs or require alternative strategies to improve musculoskeletal and cardiovascular health.

TABLES AND FIGURES

Table 1. Subject Characteristics. **V**alues for height, weight, fat free mass, bone density, fat mass, body fat percentage, baseline VO_{2peak}, and age for each subject Values are means ± S.E.M., n = 6 total.

Table 1. Subject Characteristics	
Age (yrs)	61 ± 2.6
Height (cm)	175 ± 4.5
Weight (kg)	81 ± 7.2
Fat Free Mass (kg)	54.4 ± 5.8
Bone Mineral Content (g)	3015 ± 283.3
Fat Mass (kg)	23.5 ± 2.5
Body Fat (%)	29.5 ± 2.5
VO_{2peak} (L/min)	2.91 ± .50

Table 2. VO_2 for each subject during ILE sprints during trials with 60s, 45s, or 30s recovery between sprints. VO_2 values were collected during 6 ILE sprints with differing recovery durations. Values are means ± S.E.M., n = 6 total.

Table 2. Subject Oxygen Consumption (L/min) During Differing Recoveries Between ILE Sprints				
		Recovery Durations Between ILE Sprints		
Subject	Sex	60s	45s	30s
BB120	M	1.58 ± 0.02	1.85 ± 0.02	1.81 ± 0.01
BB77	M	1.23 ± 0.02	1.95 ± 0.04	1.74 ± 0.02
BB99	F	0.41 ± 0.01	0.90 ± 0.01	0.86 ± 0.01
BB37	M	1.52 ± 0.02	2.28 ± 0.01	2.52 ± 0.01
BB122	F	0.83 ± 0.01	1.10 ± 0.01	1.22 ± 0.01
BB55	M	1.00 ± 0.01	1.91 ± 0.01	1.53 ± 0.01
	Mean	1.1 ± 0.18	1.67 ± 0.22	1.61 ± 0.23

Table 3. Average power maximum during 6 ILE sprints with either 60s, 45s, or 30s recovery between sprints. Values are means ± S.E.M., *n* = 6 total.

Table 3. Power Maximum (Watts) During 6 ILE Sprints

		Recovery Durations Between ILE Sprints		
Subject	Sex	60s	45s	30s
BB120	M	1152.00 ± 13.78	1261.00 ± 14.97	1233.00 ± 10.88
BB77	M	810.80 ± 6.35	785.20 ± 7.57	831.70 ± 8.63
BB99	F	384.80 ± 5.24	459.30 ± 6.52	440.20 ± 14.18
BB37	M	860.00 ± 13.14	932.70 ± 14.31	998.00 ± 9.30
BB122	F	670.80 ± 7.91	560.80 ± 8.61	638.00 ± 5.00
BB55	M	830.50 ± 11.01	829.20 ± 15.24	820 ± 12.84
	Mean	784.70 ± 38.98	804.80 ± 44.12	826.90 ± 42.71

Table 4. %HR maximal for each subject averaged between the end of the second and fifth sprint of 6 ILE sprints during trials with 30s, 45s, or 30s recovery between sprints. Values are in means ± S.E.M., n = 6 total.

Table 4. %HR Maximal Averaged During ILE Sprint Trials				
		Recovery Durations Between ILE Sprints		
Subject	Sex	60s	45s	30s
BB120	M	55.98 ± 0.54	58.70 ± 0.50	61.14 ± 0.82
BB77	M	63.54 ± 0.55	73.20 ± 0.83	76.8 ± 2.76
BB99	F	73.18 ± 0.56	79.33 ± 2.24	84.36 ± 1.68
BB37	M	71.35 ± 2.16	75.68 ± 0.55	79.73 ± 1.90
BB122	F	63.69 ± 0.60	85.42 ± 8.61	73.21 ± 0.60
BB55	M	72.16 ± 2.27	78.98 ± 1.14	75.85 ± 1.42
	Mean	66.65 ± 2.75	75.22 ± 3.71	75.18 ± 3.21

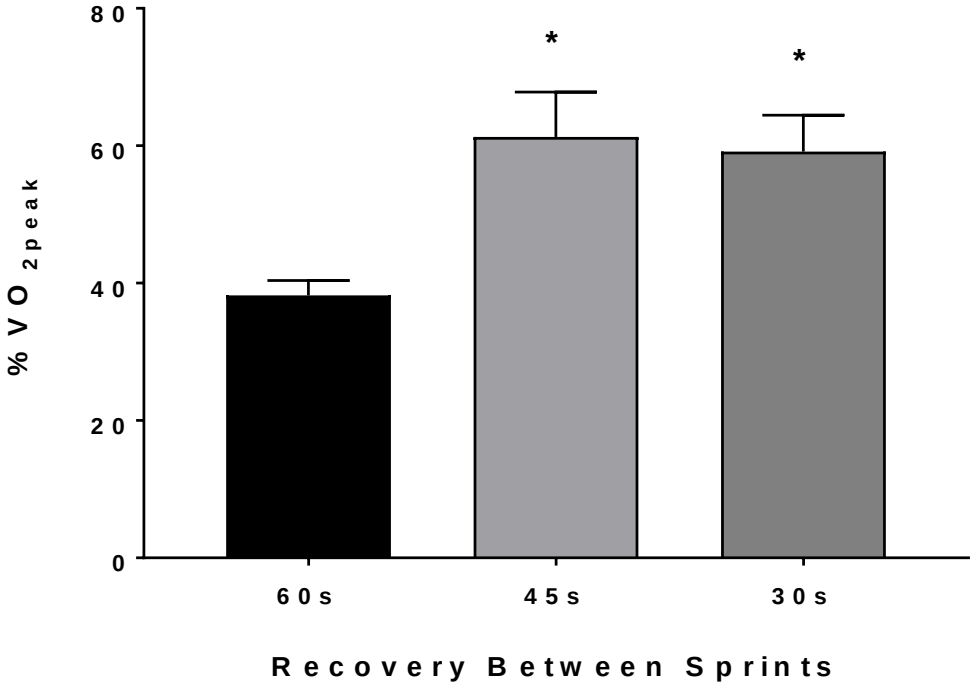

Figure 1. %VO_{2peak} during repeated ILE sprint sessions with recovery durations of 60, 45, and 30s recovery between sprints. Subjects (n=6) VO_2 was measured while on the inertial load bike for 6 ILE sprints during sessions with 60s, 45s, and 30s recovery durations between sprints. Error bars represent standard error of the means. Symbols indicate a significant difference compared to the session with 60s recovery (*) ($p < 0.05$).

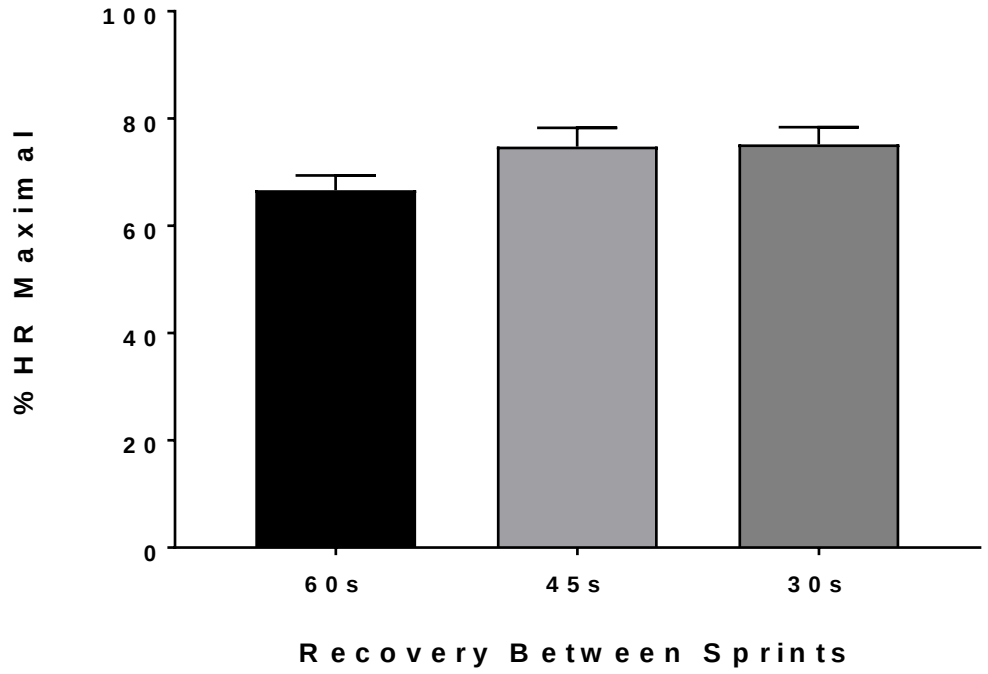

Figure 2. %HR maximal during repeated ILE sprint sessions with recovery durations of 60, 45, and 30s recovery between sprints. Subjects (n=6) VO_2 and heart rates were measured while on the inertial load bike for 6 ILE sprints during sessions with 60s, 45s, and 30s recovery durations between sprints. Error bars represent standard error of the means. There was no significance between trials ($p > 0.05$).

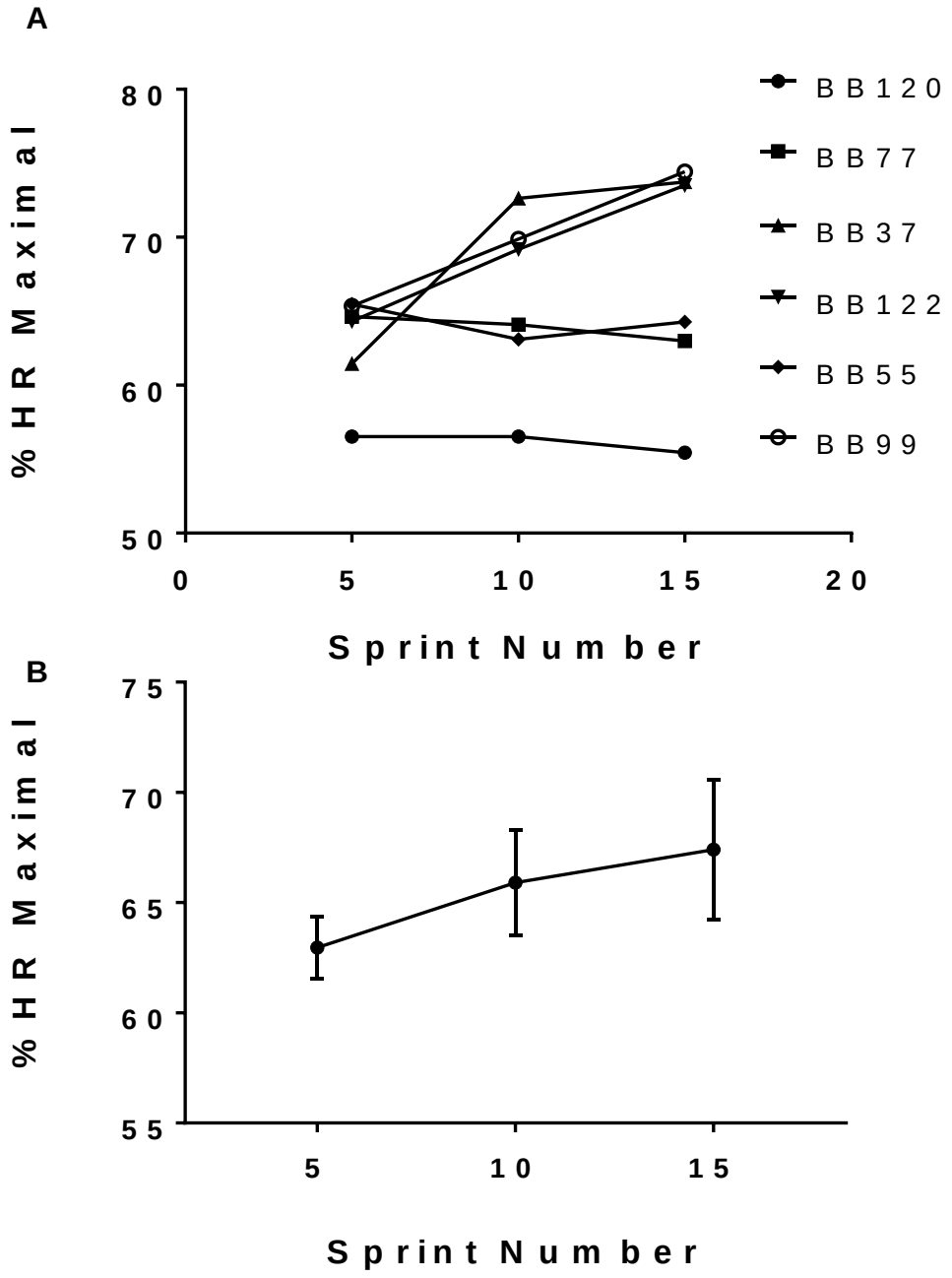

Figure 3. %HR maximal during repeated ILE sprint sessions with 60s duration recoveries. Subjects performed 15 ILE sprints with 60s recoveries during week 1 of an 8-week training program with heart rates recorded every fifth ILE sprint. Points represent the mean responses. Error bars represent standard error of the means. No significant differences were found between time points ($p > 0.05$).

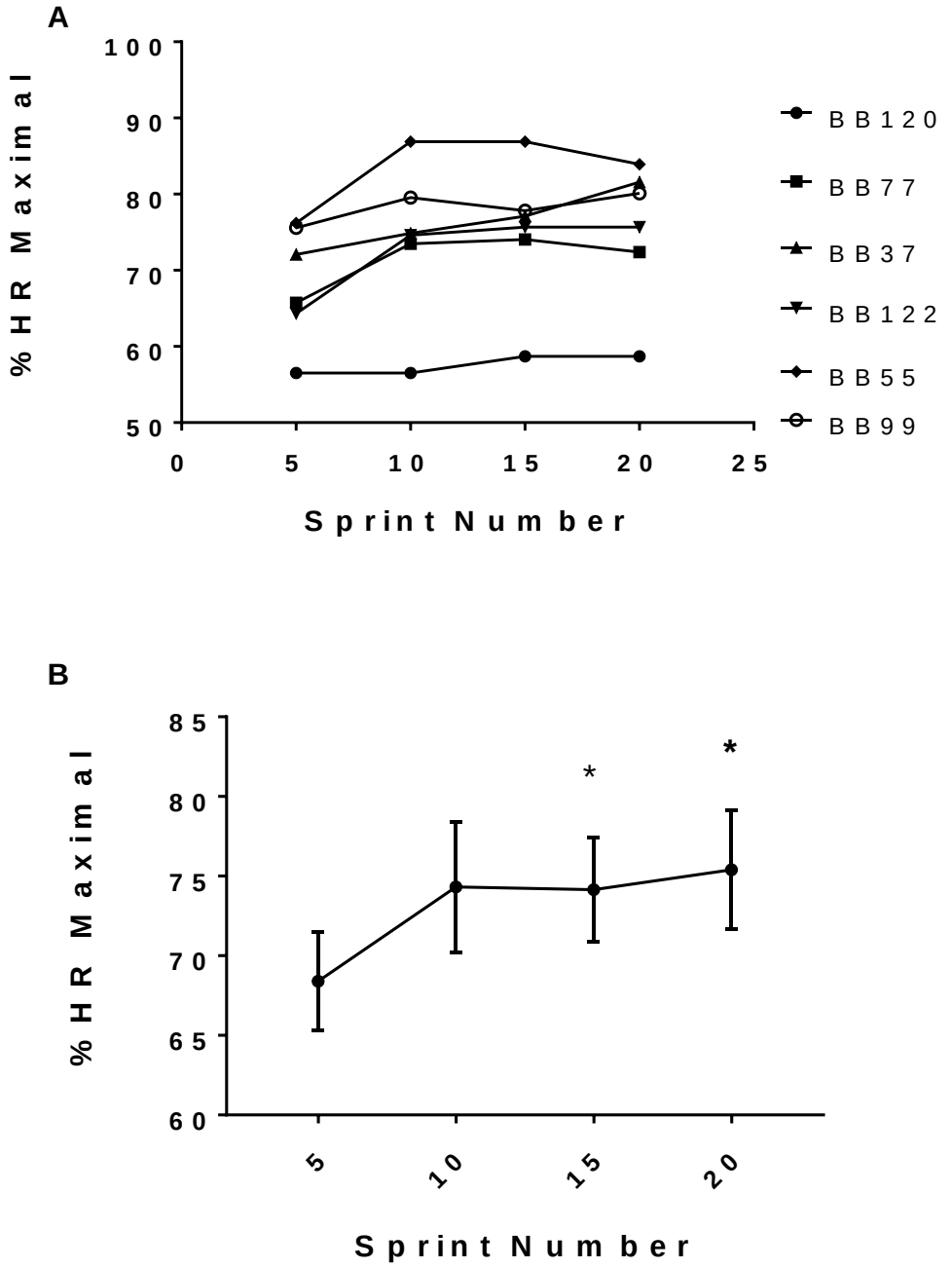

Figure 4. %HR maximal during repeated ILE sprint sessions with 45s duration recoveries. Subjects performed 20 ILE sprints with 45s recoveries during weeks 2-4 of an 8-week training program with heart rates recorded every fifth ILE sprint. Points represent the mean responses. Error bars represent standard error of the means. There was was a significant difference between group means as an effect of time compared to sprint 5 (*) ($p < 0.05$).

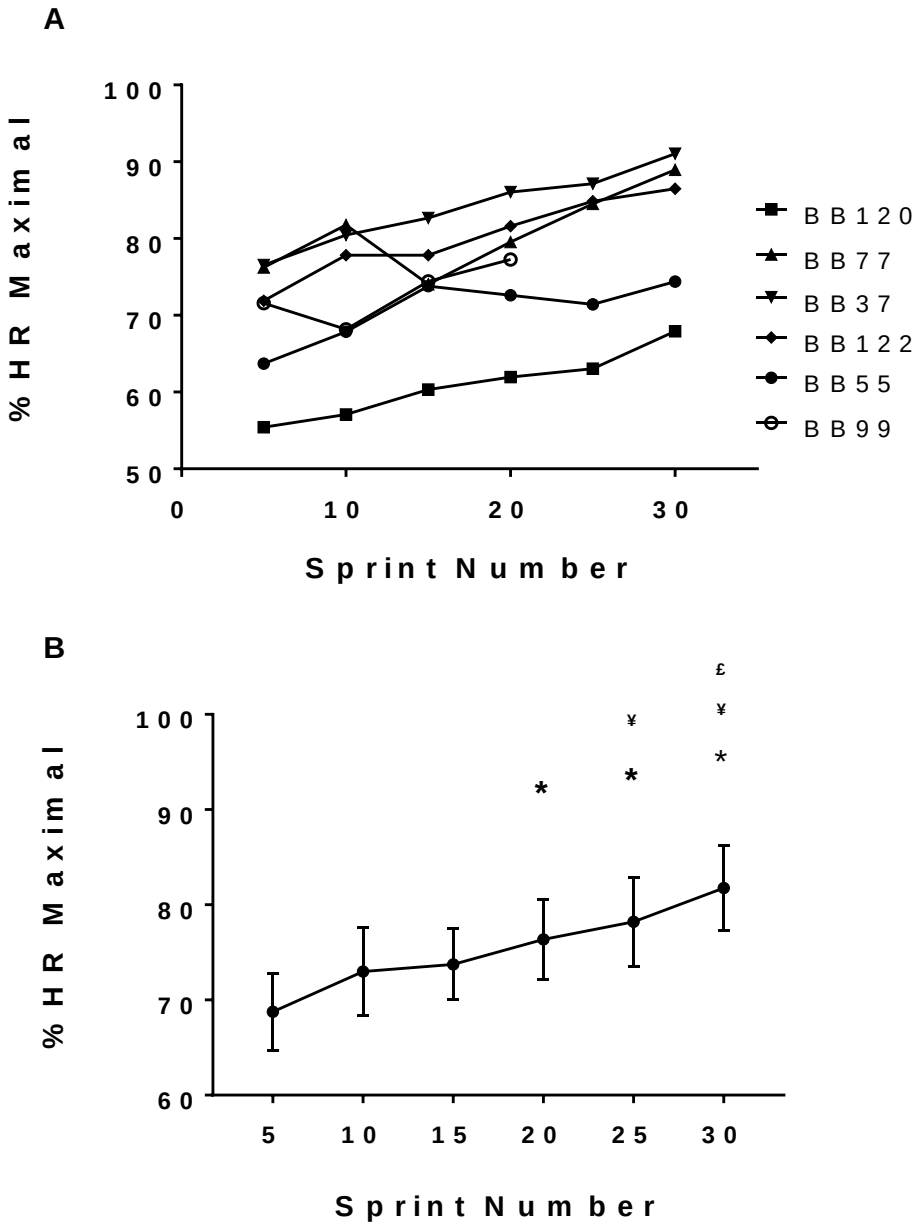

Figure 5. %HR maximal during repeated ILE sprint sessions with 30s duration recoveries. Subjects performed 30 ILE sprints with 30s recoveries during weeks 5-8 of an 8-week training program with heart rates recorded every fifth ILE sprint. Points represent the mean responses. Error bars represent standard error of the means. There were significant differences between group means as an effect of time compared to sprint 5 (*), sprint 10 (¥), and sprint 25 (£) ($p < 0.05$).

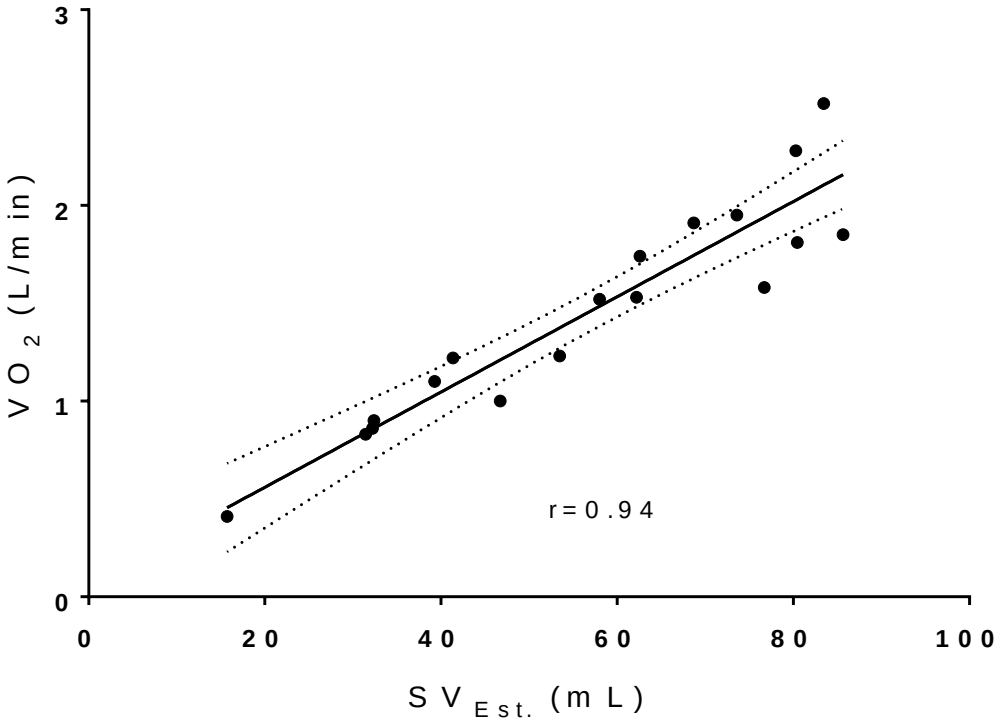

Figure 6. Correlation of estimated stroke volume vs. VO_2 during trials. Using the equation, $SV_{Est.} = 5 \text{ X } \Delta VO_2/\Delta HR$, VO_2 drives the value of the estimated stroke volumes as indicated. Dashed lines represent the bands of a 95% confidence interval. There is a strong correlation between both variables ($r=0.94$; $p < 0.05$).

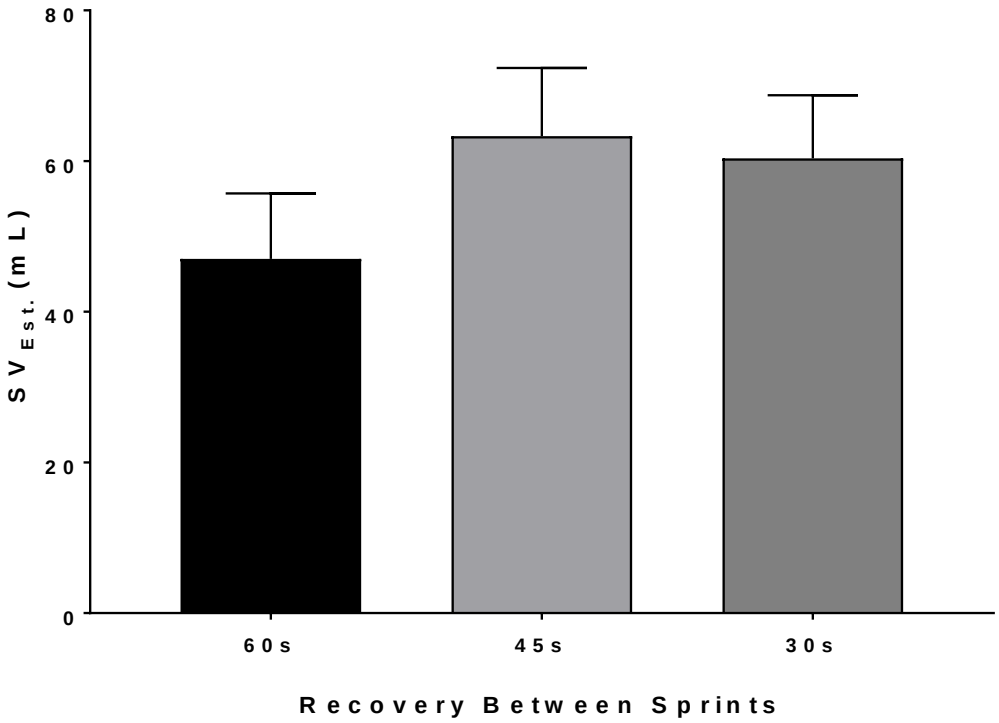

Figure 7. Estimated stroke volume during repeated ILE sprint sessions with recovery durations of 60, 45, and 30s recovery between sprints. Subjects (n=6) VO_2 and heart rates were measured while on the inertial load bike for 6 ILE sprints during sessions with 60s, 45s, and 30s recovery durations between sprints. Using O_2-Pulse and a constant, using the formula, ((VO_2/HR) x 5), a stroke volume estimate ($SV_{Est.}$) was calculated for each subject during each trial. Error bars represent standard error of the means. No significant differences were found between groups ($p > 0.05$).

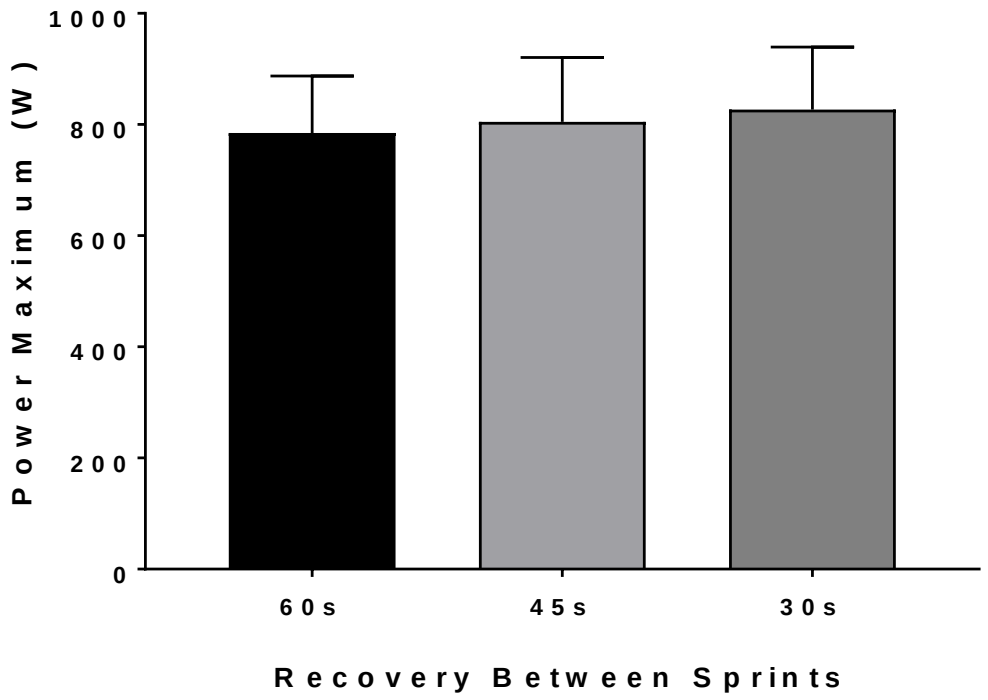

Figure 8. Average power maximums during repeated ILE sprint sessions with recovery durations of 60, 45, and 30s recovery between sprints. Subjects (n=6) VO_2 and heart rates were measured while on the inertial load bike for 6 ILE sprints during sessions with 60s, 45s, and 30s recovery durations between sprints. Error bars represent standard error of the means. There were no significant differences between trials ($p > 0.05$).

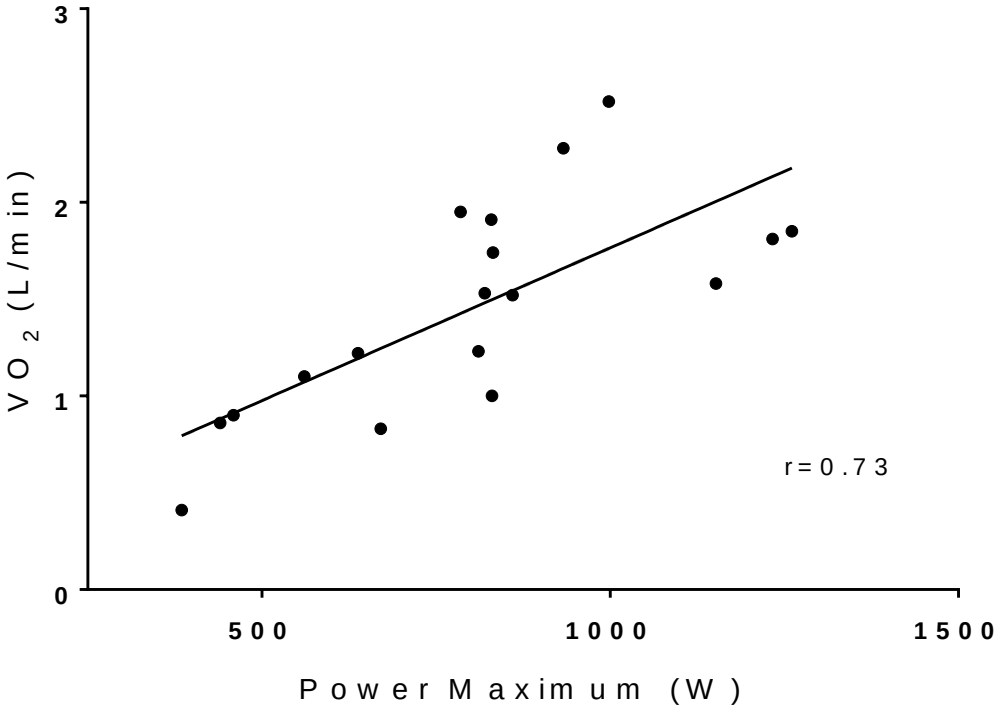

Figure 9. Correlation of power maximum vs. VO_2 during repeated ILE sprints in sessions with recovery durations of 60, 45, and 30s recovery between sprints. Subjects performed 6 ILE sprints during each trial. There is a strong correlation between both variables ($r=0.73$; $p < 0.05$).

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